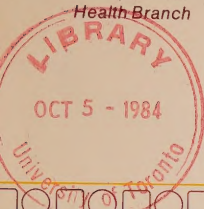




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Occupational Health in Ontario

Volume 1, Number 3 July, 1980

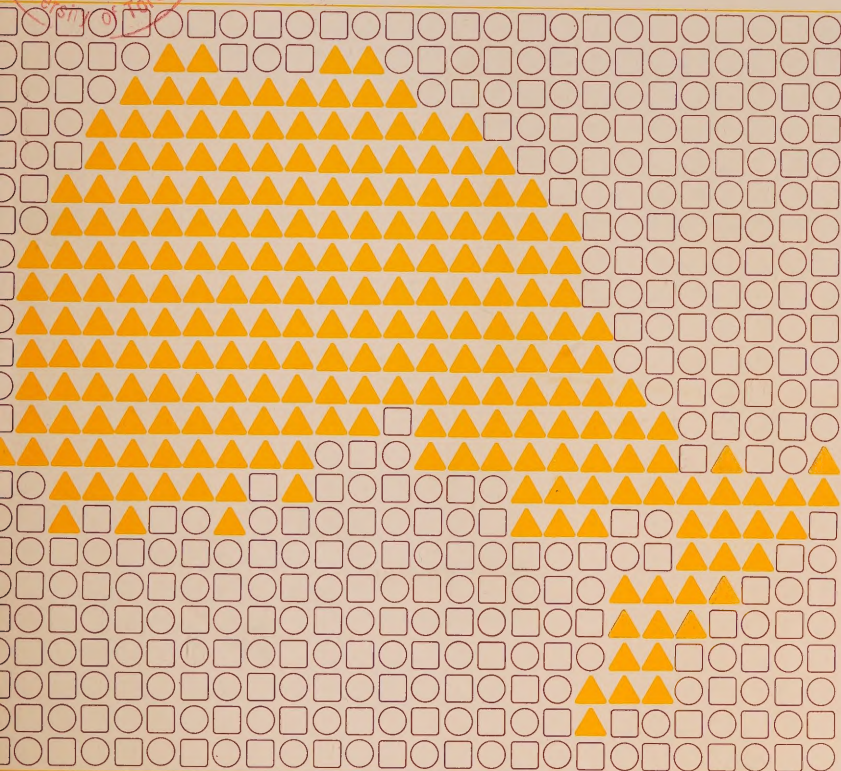
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JUL 1980

Acrylonitrile

*Medical Surveillance –
Lead Workers*

*Lead and Its Effects on
the Health of Workers*

*The Occupational Health
and Safety Resource
Centres in Ontario*



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Contents

S.S. Guirguis,
MD, DIH

Acrylonitrile 2

Jean O'Heany,
MD

Lead and Its Effects on the Health of Workers 11

G. Debow,
MD, DIH

Medical Surveillance -
Lead Workers 27

R.E.M. Lees,
MB, ChB, MFCM, DPH

The Occupational Health and
Safety Resource Centres in Ontario 30

Book Review 34

News and Notes 35

Coming Events 42

Occupational Health in Ontario

Volume 1, Number 3 July, 1980

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1. Lee WR, and Miles S. Postgraduate preparation for occupational medicine. *J. Soc. Occup. Med.* 1976; 26:115-119.
2. Department of Health and Social Security. Notes on the diagnoses of occupational diseases, rev ed 1972; London HMSO.
3. Gamble JF, and Battigelli MC. Epidemiology. In: Clayton GD and Clayton FE. eds *Patty's industrial hygiene and toxicology*. New York: McGraw Hill, 1978: Vol 1; 113-116.

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Summary

The toxicity, uses, occupational exposure, handling procedures and quantities of acrylonitrile used in Ontario are described. Guidelines are given for the necessary precautionary measures and engineering controls to prevent exposure.

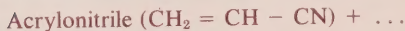
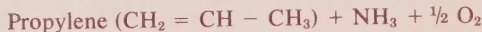
Acrylonitrile (synonyms: vinyl cyanide, cyanoethylene, propenenitrile) is also known as "Acrylon", "Carbacryl", "Fumigrain", "Ventrox".

Acrylonitrile (AN) is a chemical widely used in the manufacture of acrylic fibres, synthetic rubbers and plastics. It is a colourless liquid having a mild odour resembling peach seeds with a molecular weight of 53.06. It is very reactive and polymerizes violently in the presence of strong bases. Polymerization inhibitors are needed during storage because pure acrylonitrile may polymerize spontaneously, particularly in the absence of oxygen or on exposure to light.

It is a volatile (vapour pressure 83 mm Hg at 68°F) flammable liquid with a flash point of 30°F (open cup), is easily ignited and may decompose to free cyanide when burned. Its vapours are heavier than air and it is explosive over a considerable range of concentrations in air (3 to 17% by volume).

Manufacture and Use

AN is produced almost exclusively by the ammoxidation of propylene in the presence of a catalyst:



Production is extensive in the United States, Europe and Japan (see Table I).

Approximately 755,000 tons of AN were produced in the United States in 1975. This is expected to double by 1981. As illustrated in Table II, the major uses are:

- a) *Acrylic fibres.* More than half of AN production goes into acrylic fibres used mainly in clothing and textile industries.
- b) *Copolymer resins of plastics,* such as the resins ABS (Acrylonitrile-Butadiene-Styrene) and SAN (Styrene-Acrylonitrile). These are used for pipe and fittings, automotive parts, appliances, furniture, building components, etc.

- c) *Nitrile rubbers* which are resistant to chemicals, oils, solvents, heat, aging and abrasion. These are mainly used for industrial purposes, and
- d) *Miscellaneous* uses in production of acrylamide, acrylic esters, adhesives, adiponitrile, alkyd resins, antioxidants, coatings, cyanoethylated nature fibres and paper, dielectric paper, dyes, electrically conductive rubber, emulsifying agents, insecticides, latex paints, photographic emissions, plasticizers, synthetic leathers, wire insulation, floor polish and inks.

AN is not presently produced in Ontario. Table III shows the supply pattern to plants.¹ These plants use the monomer in amounts ranging from about 20 million lb to about 6.5 million lb per year per company. So far, 37 plants have been identified as users or possible users of AN in Ontario.

Toxicity

Inhalation of acrylonitrile causes eye irritation, sneezing, light-headedness, headache, nausea, vomiting, diarrhea and weakness. Skin contact with acrylonitrile can cause blistering of the skin after several hours of no apparent effect.

Exposure to high concentrations may produce profound weakness, asphyxia and death. It is a cyanide-releasing compound, and its acute toxicity was thought to be due to cyanide poisoning.^{2,3} However, Magos⁴, and subsequently Hashimoto and Kai⁵ on the basis of animal studies, concluded that the toxicity was due largely to the compound and not to liberated cyanide. Animal studies showed that AN affected the central nervous system, lungs, liver and kidneys.

In May 1977, E.I. du Pont de Nemours and Company Inc. informed NIOSH of the results of a preliminary epidemiologic study demonstrating an increased incidence of cancer, especially in the lung and large intestines, among workers exposed to acrylonitrile at a Du Pont textile plant in Camden, South Carolina.^{6,7}

Their followup report in 1979⁸ emphasized the excess of respiratory cancer (8 as compared with 4.4). Other factors *viz* mixed chemical exposures and cigarette smoking could not be evaluated.

In April 1977, NIOSH received from the Manufacturing Chemists Association (MCA) a one-year interim report of ongoing ingestion and inhalation studies of acrylonitrile in laboratory rats; the rats developed a

variety of tumors, including malignancies.⁹ It may be relevant to note that the chemical composition of AN resembles that of vinyl chloride monomer (VCM).

These epidemiologic findings and the experimental studies indicate that AN must be handled in the workplace as a suspect human carcinogen.

Permissible Exposure Levels

The American Conference of Governmental Industrial Hygienists (ACGIH), and the U.S. Occupational Safety and Health Administration (OSHA) prior to the recent findings were adopting 20 ppm as a TLV (TWA) for acrylonitrile exposure.

NIOSH, in September 1977, recommended that exposure to ACN be limited to 4 ppm or 8.7 mg/m³ of air as determined by a four-hour sample.¹⁰

OSHA, in January 1978⁷ adopted a temporary standard of 2 ppm averaged over an eight hour period with a ceiling of 10 ppm for any 15-minute period during eight hours.

A permanent standard proposal of OSHA considered the following options TWA 2 ppm – ceiling 10 ppm; TWA 1 ppm – ceiling 5 ppm and TWA 0.2 ppm – ceiling 1 ppm. In October, 1978, the temporary standard was adopted as permanent.

The ACGIH in 1979 listed AN as a substance with recognized carcinogenic potential without an assigned TLV, thus recommending no exposure or contact by any route – respiratory, skin or oral as detected by the most sensitive methods.

Occupational Exposure

About 10,000 workers in the U.S. handle acrylonitrile monomer directly and they may be the most exposed. NIOSH estimates that there are a total of 125,000 workers with some exposure to acrylonitrile in other industries. Over 600 workers are engaged in raw materials handling industries in Ontario.

Inhalation of vapour or skin contact may occur in many work situations, e.g., when unloading pure monomer from tank cars; during cleaning and maintenance of reactor vessels; from leaks in process equipment, during coating processes, drying processes, mixing and formulating operations, extrusion and heating of acrylic resins and the spinning of acrylic fibres.

In synthetic fibre production, employee exposure to AN is greatest during the wet spinning process since residual monomer in the fibre is released with the

solvent. There is a negligible exposure to AN of persons handling the formed acrylic fibres. This is because residual AN levels are usually well below 20 parts per million for all finished fibres, and the AN is not released easily.

Acrylic fibres tend to cyclize at temperatures above 250°C instead of reverting to their component monomer, so little if any acrylonitrile exposure results from decomposition of the fibres.

During the ABS and nitrile rubber manufacturing processes, exposure to AN may occur from leaks in equipment, openings in the system and from the residual monomer. When the intermediate product is an AN polymer resin, such as ABS, SAN, nitrile rubber and others, there is a possibility of AN exposure of employees involved in the processing of the resin into a final product (by the release of the residual AN) during any of the thermoplastic conversion processes, such as injection moulding, extrusion, blow-moulding or cold stamping of sheet plastic, etc. In general, the higher the residual AN content in the polymer, the higher the resultant level of AN vapours released into the workplace during processing.

A further source of AN exposure is connected with ABS and other AN resins during the phenomenon of "depolymerization". When these resins are heated to temperatures well in excess of their melting points, the polymers tend to revert to their component monomers. Although depolymerization would most likely occur because of an accident, the possibility of its occurrence in situations involving heat-processing must be considered.

Surveillance

A. Work Environment

Hygiene practices to reduce exposure to acrylonitrile should include:

1. Engineering controls, such as process enclosure or local exhaust ventilation to keep concentrations of airborne acrylonitrile below the recommended limit. Because of the explosive potential of AN, spark proof ventilation systems should be selected.
2. Double mechanical pump seals on polymerization operations to reduce leakage, and effective stripping of the AN monomer before the polymer is dried and blended to decrease exposure at the processing stages.

3. Isolation of operations involving acrylonitrile to reduce exposure of employees not directly involved with acrylonitrile operations.
4. Restricted access except to persons who have been properly informed of the potential hazards of AN and the proper control measures.
5. Good personal hygiene. Employees should be required to wash all exposed areas of the body upon exiting from work areas, and to shower at the end of the work shift.
6. Good housekeeping practices to prevent contamination of areas and equipment with liquid acrylonitrile. Vacuum cleaning, or any method which does not increase the airborne concentration of AN should be used.
7. Storage of containers in a safe manner to minimize accidental breakage. Spills should not come in contact with strong oxidizers (especially bromine), strong bases, copper, copper alloys, ammonia and amines.
8. Disposal of waste material in a manner acceptable to the Ministry of the Environment regulations.
9. Procedures for emergencies, including fires. Emergency equipment should be kept in accessible places.

B. Personal Protection

1. Respirators should only be used as an interim measure while engineering controls are being installed, during maintenance and emergencies. The appropriate respirators are those supplying positive air pressure.
2. Coveralls or similar full-body protective clothing and shoe coverings should be worn by employees entering work areas. Upon exiting from these areas, the protective clothing should be placed in a suitably marked and closed container for disposal or laundering. Laundry personnel should be made aware of the potential hazard in handling contaminated clothing. Leather should not be used as it is readily penetrated. Rubber should be frequently washed and inspected because it will soften and swell.

C. Medical Surveillance

A medical supervision program should be established for workers at risk. Medical examinations to detect toxic and/or carcinogenic effects of AN are necessary.

The following is suggested:

1. *Pre-placement Examination*

Exclude those with a history of fainting spells or convulsive disorders, as this will be an added risk to workers working with toxic nitriles.

The medical examination should especially concentrate on skin, respiratory tract, heart, CNS, renal and liver function. (The possible target sites for cancer include respiratory system and gastrointestinal system).

Additional tests should include

- Chest X-rays PA 14"×17"
- Liver function tests
- Fecal occult blood on all workers 40 years of age or older, and others if considered necessary.

The baseline health status will allow the detection of toxic/or carcinogenic changes during periodic examinations.

2. *Periodic Examination*

This should be similar to the pre-placement examination. Symptoms related to exposure should be included in the medical history. The examinations should be done annually or earlier if clinically indicated.

First Aid

1. *Skin and Eye Contact*

- a) Remove from contaminated area.
- b) Wash the skin promptly with abundant soap and water, or irrigate the eye with water for 15 minutes.

2. *Unconsciousness*

Emergency treatment kits containing 0.2 mL (3 minims) pearls or ampules of amyl nitrite, 10 mL of 3% sodium nitrite and 50 mL of 25% sodium

thiosulphate with suitable syringes and needles should be immediately available.

A physician should be called. Emergency measures in the event of unconsciousness in acute over-exposure should follow those for cyanide poisoning.¹¹

- a) Remove to uncontaminated atmosphere.
- b) Give amyl nitrite inhalation, 1 pearl (0.2 mL) every five minutes up to a maximum of six. Stop administration if the systolic BP goes below 80 mm Hg.
- c) Give artificial respiration with 100% oxygen in order to maintain high blood oxygen tension.
- d) Sodium nitrite 3% solution 10 mL I.V. at a rate of 2.5-5 mL/min may be used instead of amyl nitrite. Administration of nitrite forms methemoglobin, which combines with cyanide to form cyanmethemoglobin. The amount of nitrite administered must be based on hemoglobin level and on the weight of the individual.

Further administration of nitrites should be based on methemoglobin determinations, and the total methemoglobin should not exceed 40%.

- e) Sodium thiosulphate 25% I.V. at a rate of 2.5-5 mL/min should follow the nitrite therapy to convert cyanide to thiocyanate.

Cobalt EDTA may be used as an alternative I.V. therapy¹² in selected cases.

TABLE I

**Total Installed World Acrylonitrile Capacity,
Year End, 1975***

<i>Country</i>	<i>Production capacity 1000 Metric Tons</i>
United States	755
Federal Republic of Germany	330
Japan	731
U.S.S.R.	125
United Kingdom	160
Others	642
Total	2743

*Kirk-Othmer Encyclopedia.

TABLE II**The Acrylonitrile Usage – Percentages Distribution for 1975, 1970 and 1965***

<i>Use</i>	<i>1975</i>			<i>1970</i>			<i>1965</i>		
	<i>U.S.A.</i>	<i>Europe</i>	<i>Japan</i>	<i>U.S.A.</i>	<i>Europe</i>	<i>Japan</i>	<i>U.S.A.</i>	<i>Europe</i>	<i>Japan</i>
Synthetic fibres, %	55	81	54	56	83	71	52	75	80
ABS/SAN, %	20	8	11	16	11	15	9	12	10
Nitrile rubbers, %	5	5	1	6	3	3	6	7	3
Miscellaneous, %	15	6	19	11	3	11	11	6	7
Export, %	5		15	11			22		

*Kirk-Othmer Encyclopedia

TABLE III**Supply Pattern to
Ontario Industries¹**

	<i>Weight Kilotonne</i>
1971	13
1976	23.8
1977	21.8
1978	21.8
1983 (Projection)	24.6

Acknowledgements

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*Requests for reprints should
be addressed to:*

*Dr. S. Guirguis
Medical Consultant
Occupational Health Branch
Ontario Ministry of Labour
400 University Avenue, 7th Floor
Toronto, Ontario, M7A 1T7*

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¹CPI – Product Profile – Acrylonitrile – CORPUS

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Lead and Its Effects on The Health of Workers

Summary

This paper reviews the properties, distribution and metabolism of inorganic lead. The health effects on adults are discussed. For the purposes of this paper, inorganic lead includes metallic lead (Pb), all its inorganic compounds (e.g. lead oxides and inorganic lead salts) and organic lead salts such as lead soaps. The toxicity of some organic lead compounds such as tetraethyl lead and tetramethyl lead should be considered separately.

Lead has been mined and manufactured by man for more than 2,000 years and the harmful effects of lead on the health of man have been observed for almost as long.

Because of its useful physical and chemical properties, lead is the most widely used non-ferrous metal. Industrial growth over the past two centuries has led to an increasing demand for lead and it is now estimated that there are over 100 different occupations in which there may be a hazardous exposure to lead. Since 1900, improved occupational health supervision and efforts to minimize exposure to lead have greatly reduced the incidence of severe lead poisoning. During the past decade there has been increasing concern over the health hazards associated with low level lead exposure and what are frequently referred to as the subclinical effects of lead.

Physical and Chemical Properties

Lead is a bluish or silver-grey metal which tarnishes (oxidizes) in moist air. It is very soft and malleable and is easily cast, moulded or extruded.

Atomic number	82
Atomic weight	207.21
Specific Gravity	11.34
Melting Point	327.5 deg. C.
Boiling Point (at atm. press.)	1740.0 deg. C.

Distribution

Lead occurs principally as a compound of two isotopes: Pb206 and Pb208, and is ubiquitously distributed in the earth's crust. The average concentration in the igneous rocks of the earth's continents is 12 ppm to a depth of about 30 km. The soil film contains an average of 16 ppm.¹ Uncontaminated natural waters contain less than 0.1 ppm lead, and sea water has been reported to contain from 0.08 ppm to 8 ppm.² In industrial areas where lead is mined, refined and smelted or where coal is burned, or near highways, the lead content in soil may reach several thousand ppm.

Exposure

Man's exposure is from food, drinking water and air; in any case of increased lead absorption or lead poisoning one of these is usually the main source and, although the others are still present and contribute to the total amount of lead absorbed, their contribution is less significant.

Mining, smelting and refining, as well as the manufacture of lead-containing compounds and goods, can give rise to a lead hazard. The manufacture of electric storage batteries is responsible for the largest consumption of lead, using both metallic lead in the form of lead antimony alloy and lead oxides. Lead alloys are used for cable sheathing, solder, bearing metals, brasses, type metals, collapsible tubes, noise and ionizing radiation shields. Lead is used in the construction and ship building industries, and lead pigments are still used in some paints.

The mere presence of lead in the workplace does not produce lead poisoning. The hazard of lead poisoning is determined by a number of factors which lead to the occurrence of higher or lower concentrations of respirable lead fumes or lead-containing dust particles. The nature of the process is important – high processing temperatures with fume, dust or aerosol formation and primitive working methods increase the risk of poisoning; the more extreme the conditions, the greater the risk. The adequacy of the ventilation system employed plays a part in determining the risk of lead poisoning, as does the general hygienic level of the workplace and the personal hygiene of the individual worker.

Metabolism of Lead

a) Absorption

The absorption of lead is not solely dependent upon the amount presented to the portals of entry per unit of time. Absorption is influenced by the physical and chemical state of the metal and by the age and physiological status of the subject.

Lead is absorbed through the respiratory and gastrointestinal systems. Skin absorption of lead is negligible.

The proportion of lead absorbed through the respiratory tract is dependent upon particle size, solubility, respiratory volume and physiologic individual variation. Studies of airway deposition and clearance of lead in man have not yet provided a clear indication of the daily absorption to be expected under realistic conditions. A crude estimate of the percentage of inhaled lead retained would be 30 to 40%.

Consideration of the factors which influence pulmonary absorption of lead is not only of theoretic interest but aids in the understanding of why a given concentration of lead in air may produce different blood lead levels in exposed people.

Gastrointestinal absorption is small in occupational exposure when compared with respiratory absorption. Gastrointestinal absorption in adults is believed to be 5 to 15% of ingested lead. Increased dietary protein is believed to increase gastrointestinal lead absorption. Dietary deficiency of calcium, phosphorus, iron or copper is also believed to increase lead absorption.

b) Distribution and Retention

Following the intake of lead through the gastrointestinal and respiratory systems, it reaches the circulatory system and is distributed throughout the body. Lead circulating in blood is mainly found in the erythrocytes. The lead content of body fluids and soft tissues reflect recent and current exposure, whereas the skeleton is a lead depot which reflects long-term cumulative exposure.

c) Elimination

The major portion of excreted lead appears in urine and feces. However, it is also excreted in hair, sweat, nails, breast milk and exfoliated skin.

Fecal excretion is the largest single route of elimination of ingested lead. Fecal lead is the lead which has passed through the gut without being absorbed, plus the lead which has been excreted in bile and is lost in the various intestinal secretions or by the shedding of epithelial cells.⁴ The elimination of lead which has been absorbed through any portal of entry is chiefly via the urine. The renal excretion of lead involves more than filtration of the metal at the glomerulus but the exact mechanisms are still controversial.^{37, 40}

Health Effects of Lead on Man

Acute lead poisoning is very rare in adults today; however, chronic lead poisoning still exists and, depending on the extent and duration of exposure, the effects may range from lead colic to encephalopathy and death. The classic picture of chronic lead poisoning in adults includes abdominal colic, pallor,

headache, fatigue, gum line and peripheral neuropathy. Chronic lead nephropathy may be found associated with prolonged exposure.

The toxic effects of lead vary from minimal changes, which are detectable only through laboratory examinations, to the severe clinical picture described above. Toxicity usually manifests itself in one of the target organs of the body. The "critical organ" is the most sensitive one, the organ where the earliest signs of damage are detected. The bone marrow is believed to be the critical organ in lead poisoning,⁶ although some authors suggest that the central nervous system may be the critical organ under certain circumstances.⁷ The most significant health effects of lead are found in the hemopoietic, the nervous, the renal and the reproductive systems.

Hemopoietic System

There are a number of reactions in hemesynthesis which are interfered with by lead, and it is the detection and measurement of changes produced by this inhibition, as well as blood lead levels, which serve at this time as a basis for monitoring lead effect in those exposed. The exact significance of these changes is not known; however, the fact that changes occur may in itself be significant.

The hematological effects of lead can be attributed to (i) the inhibition of hemoglobin synthesis; (ii) the shortened life span of circulating erythrocytes and (iii) the stimulation of erythropoieses. The result of these effects is anemia.

Lead interferes with hemesynthesis through inhibition of enzymes. It inhibits delta amino levulinic acid dehydratase (ALA-D) and ferrochelatase as well as amino levulinic acid synthetase (ALA synthetase) and coproporphyrinogen decarboxylase.

Of all the known biological effects of lead, hematological or otherwise, inhibition of ALA-D is believed to be the earliest. Inhibition of ALA-D has been reported at blood lead levels barely above normal.^{8, 13} A decrease in the level of ALA-D can be detected a short time after a worker's initial exposure to lead and, by the end of a month, levels of ALA-D may be reduced to 50% of pre-exposure values.⁵

The inhibition of ALA-D is well correlated with the level of blood lead (PbB) up to a PbB level of approximately 0.07 mg/100 mL. There is nothing to prove that partial ALA-D inhibition per se is deleterious.⁷

Urinary ALA (amino levulinic acid) and coproporphyrin excretion begin to rise when PbB exceeds 0.035-0.04 mg/100mL.

The inhibition of ferrocheletase produces an increase in the concentration of protoporphyrin IX (PPIX) in the erythrocytes at PbB levels of 0.025-0.030 mg/100 mL in children and women and at about 0.04-0.05 mg/100 mL in men. However, elevated levels of PPIX persist long after exposure has ceased and the correlation of PPIX with PbB levels is not always as close as it is for ALA-D. PPIX is sometimes referred to as FEP (free erythrocyte protoporphyrin IX).

Reticulocytosis becomes measurable when PbB is between 0.06-0.08 mg/100 mL. In this range a shortening of erythrocyte life span also begins. The degree of anemia correlates poorly, if at all, with PbB levels. A slight drop in the Hb level has been shown at PbB levels of 0.05-0.08 mg/100 mL. Obvious anemia is not usually seen below a PbB level of 0.08 mg/100 mL.

Table I.
Approximate First Detectable Levels for Toxic Effects in Blood Lead Concentrations in Adults

<i>Approx. Blood Lead concen'n. mg/100 mL</i>	<i>Toxic Effect First Detectable</i>
0.01	Erythrocyte ALA-D inhibition
0.025 (a)	Increased PPIX or FEP
0.035 (b)	Increased PPIX or FEP
0.040	Increased Urinary ALA
0.040	Increased Urinary Coproporphyrin
0.050	Decreased Hb Level
0.06-0.08	Increased Reticulocytosis
0.08	Clinical Anemia

modified from Grandjean 1978

a) females

b) males

Nervous System

Lead can affect both the central and peripheral nervous systems in man, producing encephalopathy, neuropathy and, in the opinion of some researchers, psychological changes.

Central Nervous System

Encephalopathy as a result of occupational exposure to lead is extremely rare today. The major features of encephalopathy are dullness, restlessness, irritability, headaches, muscular tremor, hallucinations, loss of memory and inability to concentrate. These signs and symptoms may progress to delirium, mania, convulsions, paralysis and death. The onset and course of lead encephalopathy are always unpredictable, although high blood lead concentrations (usually much above 0.150 mg/100 mL) are believed to be required. Minor psychological changes – hostility, depression and general dysphoria – have been reported among lead workers (blood lead levels of 0.070 mg/100 mL or greater). Whether these psychological changes are caused by lead or other environmental factors has not yet been conclusively demonstrated.¹⁴ Changes in sensory-perceptual and psychomotor function due to low-level lead exposure have been observed in some lead workers at PbB levels above 0.070 mg/100 mL.¹⁶

Although the psychological and performance tests utilized by these workers have been verified, the results must be shown to be repeatable in other laboratories before a dose-response conclusion can be made.⁷

Peripheral Nervous System

Inorganic lead has toxic effects on the peripheral nervous system. Neuropathy is the disease most commonly associated with lead toxicity. The typical picture is wrist drop of the right hand. However, other muscles of the upper limbs, the extra-ocular eye muscles and the extensors of the lower limbs may be involved. This picture is characterized by the selective involvement of motor neurons with little or no sensory abnormality. Neuropathy is seen rarely now and only in conditions of uncontrolled exposure.

Subclinical forms of neuropathy are of great interest. Using electrophysiological techniques it has been possible to demonstrate neurological damage in lead workers in whom no clinical damage could be shown.

Reduced nerve conduction velocity in lead workers with no overt signs of lead toxicity was reported by Catton *et al*¹⁵ Work done by Seppalainen and Hernberg,¹⁷ showed reduced motor conduction velocity of the slower fibres of the ulnar nerve in lead workers (PbB levels of 0.08-0.12 mg/100 mL) in cases with no clinical findings. Similar results were reported by Sep-

palainen in 1975 in 26 workers whose PbB levels had never exceeded 0.07 mg (exposure time 13 months to 17 years).¹⁸

The conclusions reached at that time were that lead in blood in concentrations exceeding 0.05 mg/100 mL can produce changes in nerve conduction velocity. There is no evidence that these changes progress when PbB stays stable. Reversibility needs to be studied.

Barltrop²⁹ has shown that lead can cross the human placenta and that such transfer can be demonstrated as early as the 14th week of gestation. Total fetal lead content increases as gestation progresses and is directly related to maternal lead levels. It has been postulated by Barltrop²⁹ that the fetus has increased susceptibility to the toxic effects of lead, and maternal innocuous lead intake may on occasion prove hazardous to the fetus. More critical research is needed to evaluate the subtle toxic effects of lead on the fetus.

The incidence of early membrane rupture and premature delivery among women living in a lead mining area were investigated under the direction of the Department of Obstetrics and Gynecology of the University of Missouri Medical School. Lead, copper, cadmium and zinc levels were determined in maternal and fetal blood samples and in histological samples taken from the umbilical cord, the placenta and the fetal membranes of 502 women. There were 249 women from Columbia, Missouri which was considered to be a non-lead mining area (Region 1) and 253 from Rolla, Missouri which is in the Missouri lead belt (Region 2). The patients in both regions had lived in their areas for at least 10 years, were aged 20 to 25 and in an income bracket of \$7,000 to \$10,000 annually.

The incidence of term pregnancy with early rupture of membranes was 0.41% in Region 1 and 17% in Region 2. The incidence of premature deliveries was 3% in Region 1 and 13.04% in Region 2. There was no significant difference in maternal or fetal blood lead levels in term pregnancies between the two regions. Maternal PbB in term pregnancies in Region 1 averaged 0.0131 mg/100 gm and in Region 2 0.0143 mg/100 gm. There was only one patient in Region 1 delivered at term with early rupture of membranes and her PbB was reported to be 0.0301 mg/100 gm. The group from Region 2 with term delivery and early membrane rup-

ture (17%) had an average PbB of 0.0256 mg/100 gm. Those with premature delivery in Region 1 (3%) had an average PbB of 0.0260 mg/100 gm and in Region 2 (13.04%) 0.0291 mg/100 gm (see Table 2).

The study suggests that there may be a relationship between the level of heavy metals to which the pregnant woman is exposed and the incidence of early rupture of membranes and premature delivery. The effects of lead on humans should be studied carefully from conception through puberty.

Table 2
Lead concentration in maternal blood
at time of delivery (mg/100 gm)

	<i>Term Delivery</i>	<i>Term Delivery with Early Membrane Rupture</i>	<i>Premature Delivery</i>
Region 1 (unexposed)	0.0131	0.0301 (0.41%)	0.0260 (3%)
Region 2 (exposed)	0.0143	0.0256 (17%)	0.0291 (13.04%)

adapted from reference #28

figures in brackets () refer to percentage of group affected.

Renal System

Acute

The acute effects of lead on the renal system are seen in patients dying of acute lead poisoning or suffering from lead-induced anemia and/or encephalopathy. These changes are usually non-specific degenerative changes in the proximal tubular lining cells. In the early or acute phase of lead intoxication these changes include the formation of inclusion bodies in the nuclei of proximal tubular lining cells and the development of functional as well as ultrastructural changes in renal tubular mitochondria.

Chronic

There is evidence in the literature to support a relationship between prolonged lead exposure and chronic lead nephropathy. These changes are believed to be due to prolonged exposure to excessive levels of lead.

It is believed that chronic lead nephropathy is not seen except in the presence of, or following the presence of, other more commonly described signs of lead intoxication. The duration and degree of lead exposure required to produce irreversible kidney damage is not known.

Chronic lead nephropathy is characterized by pronounced arteriosclerotic changes, fibrosis, glomerular atrophy and hyaline degeneration of the vessels. Chronic lead exposure produces two measurable effects on the renal system: (i) impairment of glomerular function producing a rise in blood urea nitrogen (BUN) and creatinine levels; (ii) abnormal renal tubular function which leads to a decrease in the kidney's ability to concentrate urine as well as to remove lead from the circulating system for excretion from the body.

The presence of intranuclear inclusion bodies is very helpful in establishing a relationship between renal lesions and lead toxicity, but inclusion bodies are not always present in persons with chronic lead nephropathy. Cramer *et al*¹⁹ reported a decreased ability of renal proximal tubular cells to form inclusion bodies and a decreased ability of the kidney to excrete lead in patients with extensive (greater than four years) exposure to elevated lead in air levels.

Reproductive System

Medical literature contains many references to the toxic effects of lead on the reproductive system and on the fetus.

Historically, women occupationally exposed to lead (and the wives of men exposed to lead) are reported to have an increased incidence of miscarriages, pregnancies ending in stillbirths and children who die in the first year of life.²⁰ More recent literature has considered that increased lead absorption may have a direct effect on ovarian²¹ and testicular function.³⁰

Several studies using animals have attempted to explain these observations in humans. Early experiments in rats showed that lead can cross the placenta.²² Lead has been shown to be fetotoxic in mice, rats²³ and hamsters,²⁴ gametotoxic in rats²⁵ and rhesus monkeys,²⁶ and teratogenic in rats²⁷ and hamsters.²⁴ The teratogenic effects which have been demonstrated

in animals are due to the use of very large amounts of lead. Present occupational exposures do not produce a comparable lead burden and therefore these results cannot be applied to humans. Human malformations attributed to lead absorption have not been reported in the literature. There is no epidemiological evidence of teratogenic or fetotoxic effects in humans.

Lancranjan³⁰ studied the reproductive ability of 150 male workers exposed to lead in a storage battery plant. Workers with moderately increased lead absorption (mean of 0.53 mg Pb/100 mL blood) showed a decrease in spermatogenesis. This observation was attributed to a direct toxic effect on the gonads which produced alterations in spermatogenesis – asthenospermia (decreased mobility) hypospermia (decreased numbers) and teratospermia (malformed sperm). Even among workers with slightly increased lead absorption (mean of 0.041 mg Pb/100 mL blood) differences in asthenospermia and hypospermia were observed.

The conclusion reached by Lancranjan *et al* was that lead had a direct effect on the germinal epithelium, since studies done on the same group of men did not show any changes in hypothalamo-pituitary axis function as measured by urinary gonadotropins and 17-ketosteroid excretion. This is a most interesting study; however, the paper did not correlate the age of the individual workmen to changes in the sperm picture but only told us the workers had a mean occupational exposure to lead of 8.5 years (1 to 23 years) and a mean age of 38½ years. These results are not conclusive and further investigation needs to be carried out.

Effects on the Endocrine System

Excessive exposure to lead has been associated with an impairment of endocrine function in man and experimental animals. Iodine uptake was decreased in lead-poisoned patients but this effect could be overcome by exogenous administration of thyroid stimulating hormone.³¹ High lead levels resulted in a decreased secretion of pituitary hormones and an impairment of adrenal gland functioning in lead-poisoned patients. Whether low-level chronic lead exposure can affect pituitary, adrenal and thyroid function has not as yet been determined.⁴¹

Carcinogenesis

There is no evidence to show that exposure to lead salts causes cancer in any site in man. Dingwall-Fordyce and Lane³² did not find any evidence of an increased death rate as a result of malignant diseases

in their study of 267 workers. The men studied retired between 1926 and 1960, all had at least 25 years of service and those in the heavy exposure category had an average urine concentration of 0.10-0.25 mg/litre (corresponding to PbB of 0.06 mg-0.12 mg/100 mL, according to Williams *et al.*³³)

Cooper and Gaffey³⁴ concluded that in a group of 7,032 men occupationally exposed to lead between 1946 and 1970, the incidence of malignant neoplasms was somewhat greater than expected, but the difference in the incidence was not statistically significant.

Teratogenesis

Animal studies (see section on reproduction) in which teratogenesis effects have been reported²²⁻²⁷ have involved very large amounts of lead. Present occupational exposures do not produce a comparable lead burden and these results should not be applied to humans.

Discussion

Chronic lead poisoning results in a well-characterized syndrome manifested in adults by colic, anemia, headache, fatigue, pallor and peripheral neuropathy,³⁵ although this syndrome is rarely seen in modern occupational medicine. Symptoms of lead poisoning are not usually seen until blood lead levels exceed 0.08 mg/100 mL.^{36, 37}

Of great concern at this time is the possibility that prolonged exposure to lower levels of lead may result in adverse health effects. Investigation has been carried out on the subclinical effects of lead, but as yet it has not been shown that the subclinical effects seen at these low blood lead levels would progress to clinical symptoms unless lead exposure and absorption were increased. There is no evidence to date that these subclinical effects are either reversible or progressive.

There is extensive literature attempting to relate blood lead levels and adverse health effects. Although adverse health effects can occur at a blood lead level of 0.08 mg/100 mL, these effects are not seen in all cases. The existence of a blood lead level of 0.08 mg/100 mL is not itself diagnostic of chronic lead poisoning.^{33, 36-38}

There is no specific number below which it is impossible to find symptoms of lead toxicity, nor above which lead toxicity is inevitable. For the protection of lead workers it is proposed that they should be removed from exposure when their blood lead level is 0.07 mg/100 mL. This level would allow a margin of

safety and should cause the worker to be removed before any disease process has begun. The decision to remove a worker from lead exposure, in those cases where a lead-related health problem is suspected, should be made by his attending physician after considering all clinical and laboratory information. This decision should not be made automatically on the basis of any single laboratory test.

The relationship between blood lead levels and air lead levels is of great interest. A research project by Williams³³ on the relationship between blood lead levels and airborne lead levels showed a range of blood lead from 0.038 mg Pb/100 mL to 0.082 mg Pb/100 mL with a mean of 0.06 mg Pb/100 mL when the workers were exposed to an airborne lead level of 0.150 milligrams per cubic meter (0.150 mg/m³). In 1964, Robert A. Kehoe³⁹ published a report outlining experiments in which volunteers were exposed to an atmosphere containing 0.15 mg Pb/m³ for 37.5 hours per week for 102 weeks. The highest concentration of lead in blood of any subject was 0.045 mg/100 gm.

Prior to 1933 the United States had an airborne exposure limit of 0.5 mg Pb/m³. In 1933, 0.15 mg Pb/m³ air was a common goal for industry. The U.S. Occupational Safety and Health Administration standard for lead and its inorganic compounds adopted in 1971 was 0.20 mg Pb/m³ air as an 8-hour time weighted average. In 1973, a request was made to lower this figure to 0.15 mg Pb/m³ air and in 1975 to 0.10 mg Pb/m³ air.

In the United States a final standard for occupational exposure to lead which reduces the permissible exposure level from 0.20 to 0.05 mg Pb/m³ air became effective February 1, 1979. A challenge to the new standard from industry has resulted in a partial stay of application pending the determination of the challenge. At the time this report was written the matter was still being considered by the courts.

In the Province of Ontario the time-weighted average occupational exposure limit for lead is 0.15 mg Pb/m³ air and the short-term occupational exposure limit for lead is 0.45 mg Pb/m³ air. From the evidence available at present there is no good reason to change these limits.

Requests for reprints should be addressed to:
Dr. Jean O'Heany,
Special Studies
and Services Branch,
Ministry of Labour,
8th Floor, 400 University Ave.,
Toronto, Ont. M7A 1T7

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Medical Surveillance – Lead Workers

Summary

This article outlines a medical surveillance program, including action levels, for lead workers in industry in Ontario.

Occupational Exposure

Lead is commonly used in industry, particularly in the battery manufacturing and lead reclaiming industries. Lead compounds are used in paint pigments, glasses, frits, plastics and rubber compounds. Exposure to any of these sources during their manufacture or in some cases their use could be a potential health hazard.

Source of Entry

Lead commonly enters the body by inhalation and ingestion. Inorganic compounds are not absorbed through the skin to any significant degree. Inhalation is the most common route of entry in industry but ingestion may occur by eating, drinking or smoking where lead is present in the work environment.

Guideline Recommended

The guideline for lead in air is 0.15 mg/m^3 . This level should protect most workers who are exposed to lead by inhalation for an eight-hour working day and a 40-hour work week.

Medical Program

The medical program should consist of a pre-placement medical examination, a periodic examination and the submission of a biologic sample. The pre-placement examination should consist of a review of previous lead exposure, a general history and physical examination, and biologic sampling for hemoglobin and blood lead. Women capable of bearing children should be made aware of the possibility of toxic exposure to the fetus through the transfer of lead from mother to child during pregnancy.

The periodic examination should usually be performed yearly but the frequency may vary with exposure and the ultimate decision should be left to the company physician. The examination should be similar to the pre-placement medical. A urine and/or blood sample should be taken for lead analysis.

Biological Samples

The frequency of submission of blood or urine for lead analysis varies with the intensity and duration of exposure. However, usually they are submitted at one- to three-monthly intervals. It is advisable to collect samples toward the end of the work week and at the start of a work shift to obtain a realistic estimate of absorption from exposure and to minimize contamination of specimens.

i) *Urine Samples*

The sample should be at least 50 mL in a lead-free plastic container with screw cap (no liner.) Add 6 drops of glacial acetic acid to specimen immediately after collection.

ii) *Blood samples*

The samples should be 10 mL of blood in a heparinized tube (or EDTA oxalate tube.) Tubes should be lead free. Whole blood is assayed.

Urine and blood samples may be sent to the Occupational Health Laboratory, 360 Christie Street, Toronto, Ontario, M6G 3C2.

Suggested Action Levels for Workers Exposed to Lead

In the industrial community there is controversy as to the level at which workers should be assessed for lead exposure and at what level changes should be made in the employees' work environment. The following are suggested action levels:

- i) When the urine lead level reaches 0.18 mg/l (specific gravity corrected to 1.025) a blood specimen should be taken for lead analysis and the worker's exposure reviewed. If the confirmed blood lead is 60 $\mu\text{g}/100\text{ mL}$ or above, the company physician should make a review of the employee's work practices, enquiring as to what improvements (e.g., personal hygiene) could be made to reduce the absorption of lead.

A health review should be done by the medical service of the company. If symptoms of lead intoxication are present then removal from work is essential.

- ii) If the confirmed blood lead is 70 $\mu\text{g}/100\text{ mL}$ or above, then the worker should be removed from his present lead exposure, and not be allowed to return to his regular employment until the blood lead level returns to 50 $\mu\text{g}/100\text{ mL}$ or less.

iii) If symptoms of lead intoxication occur, remove from exposure, regardless of level.

The company medical service staff should review all laboratory reports as they are received to detect any significant increase in results, and take appropriate action to ascertain the cause. They may wish to do other tests, e.g. ALA, coproporphyrins, to evaluate the extent of the effects of exposure. The medical and nursing consultants of the Ministry of Labour, Occupational Health Branch, are available to give further advice, if required.

<i>Location</i>	<i>Telephone No.</i>
Toronto	965-3610
Hamilton	527-2951
London	439-3231
Ottawa	523-2701
Sudbury	675-4455

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*Requests for reprints should
be addressed to:*

Dr. G. Debow

Occupational Health

Medical Service

Occupational Health Branch

Ministry of Labour

400 University Avenue

7th Floor

Toronto, Ontario

The Occupational Health and Safety Resource Centres in Ontario

Summary

The responsibilities, development and role of the four resource centres in Ontario are discussed.

In response to requests from several provincial universities and colleges for support funding of occupational health programs, the Government of Ontario promoted the establishment of four Occupational Health and Safety Resource Centres in 1978. Funding was provided through the Provincial Lottery Fund and was administered by the Ministry of Labour under whose portfolio occupational health legislation was being consolidated at that time. The grants were clearly identified as establishment funds to be used to build up facilities which would be financially independent of the original grant at the end of three years.

The four locations were selected on the basis of three major criteria: the quality of proposals submitted and the stated objectives, the availability of personnel skilled and experienced in some aspect of occupational health, and a geographic position in accordance with ministry plans for providing regional resources to industry. The last criterion might not have been ideally met but failure to achieve the ideal was determined by the other two criteria, even if a suitable institution was conveniently located within a region. It became obvious that a fourth factor probably entered into the selection equation – industrial density. Industrial density was a more realistic criterion than territorial extent of the region, since an earnings potential for each centre was imperative if long-term financial independence was to be achieved.

The centres were established at Lakehead University (Thunder Bay), Queen's University (Kingston), the University of Western Ontario (London) and Waterloo University (Kitchener). Territorially, Lakehead with north and northwestern Ontario and Queen's with the eastern region had the greatest areas of responsibility while Waterloo and Western were in areas of greater industrial concentration.

Responsibilities

In addition to developing the objectives stated in their individual proposals, the centres were charged with specific responsibilities by the terms of the grant from the Provincial Lottery Fund. These responsibilities were:

1. To undertake a survey of occupational health and safety needs of employers and employees in the region.
2. To provide a service to meet the needs of management and labour in regard to occupational health and safety.
3. To establish a directory of consultative expertise in the university and private sectors and to act as intermediaries in supplying an interface between these consultants and industry.
4. To assist educational institutions, industries and labour in setting up training programs in occupational health and safety.

An additional geographic/industrial responsibility was put upon the Queen's centre: namely, that it discharge responsibility number 1 in respect of the mining industry of northeastern Ontario.

In providing consultations and services to industry it was expected that the centres would charge fees which would contribute to their support beyond the termination of grant funding in 1981.

Development

After meeting the basic responsibilities placed upon it, each of the centres has evolved along slightly different lines from its sister institutions. The differences have not occurred haphazardly, but as a result of the special expertise and facilities at each centre and the identified needs of labour and management in the region. These specializations have been coordinated, but not directed, by the four directors of the centres, who meet officially every three months to discuss policy and progress. The general intention has been to develop a chain of resource centres across the province, each individually capable of meeting the general needs of its region but collectively able to provide specialized services and expertise to industries throughout Ontario.

There have been differences, too, in the educational and academic activities of each of the centres. Only the Queen's centre has developed a major interest in undergraduate education, but all have produced continuing technical and professional educational programs designed to benefit the industrial communities within their regions and the province as a whole. Examples are symposia on the applications of Bill 70 and its regulations, industrial hazard identification, occu-

pational nursing, noise control, and short courses on monitoring the industrial environment, hearing conservation and industrial audiometry. The symposia have tended to deal with regional concerns, while the short courses have been open to persons from all parts of the province.

Some of the centres have provided opportunities for elective attachments by persons wishing to acquire extra knowledge of, or skills in, occupational health and safety practice – laboratory technicians at Western and medical technicians at Queen's.

The Lakehead centre is specializing in the problems of the pulp and paper industry, Queen's in occupational medical practice and occupational hygiene, Western in environmental hazard analysis, and Waterloo in ergonomics. Collectively, therefore, the centres now provide Ontario with a resource of skills, experience and facilities which cannot be found in any other part of the country.

Research is an integral part of resource centre interest. Support for research was not provided under the establishment grant from the lottery fund. Research funds have been obtained by the centres in the customary academic manner – by open competition from government, independent foundations and research agencies, or by contracts from individual institutions. In general, applied rather than basic research has been undertaken by the centres. That policy is likely to continue. From the group as a whole research interests are broad, but the majority of projects undertaken so far have been determined by the interests and at the initiative of individual members of staff. In the short period of the centres' existence their research record has been good.

Trade unions, individual employees, managers, physicians and nurses have made extensive use of the centres for information regarding matters of occupational health concern and advice concerning the correction of problems. Information and advisory services are recognized to be a major contribution of the centres to the industrial communities and their regions. The demand for these services is likely to increase considerably in the months ahead, as enforcement of the regulations made under the Act Respecting the Occupational Health and Occupational Safety of Workers increases. Unfortunately, that serv-

ice is the most vulnerable to the reduction in financial support which might follow the termination of the establishment grants in the spring of 1981.

There has been controversy in several quarters surrounding the establishment and the location of the centres. Controversy, at times bordering on open hostility, is probably the result of misunderstanding about the purpose of the centres and the concepts which led to their establishment. Perhaps this description of their aims, objectives and activities will clear up some of these misunderstandings. The centres must be recognized for what they are: resources to the industrial community (in the broadest definition of that term) of Ontario and an asset to the province. Their full potential in education, service consultation, research and as information and advisory centres will only be achieved if the industrial community utilizes them and supports them. Being free of ties to any partisan institution they are in a unique position to promote occupational health and safety in the province and to respond to the needs and requests of industry. Failure of these centres to continue their existence would be an irretrievable loss to Ontario.

Requests for reprints should be addressed to:

*Dr. R.E.M. Lees,
Occupational Health
Resource Centre
Health Sciences
Queen's University
25 Union Street
Kingston, Ontario
K7B 2N6*

Book Review

5th Edition - 1980
Alliance of American Insurers -
134 pp

Handbook of Organic Industrial Solvents Technical Guide No. 6

The purpose of this book is to provide the health and safety professional with fundamental information regarding the recognition and evaluation of solvent exposure hazards and the principal methods of control.

The format of the book does not differ much from the 4th edition, although the descriptive text is more organized. The list includes eight new solvents.

The book adopts the National Fire Protection Association (NFPA) hazard identification system, while recommending the Manufacturers Chemists Association labeling system for the control of solvent exposures. This reflects current confusion which will persist until a specific uniform labeling system is introduced by regulation.

The book remains a good quick reference guide; however, more detailed information may be still needed from other texts.

S. Guirguis

Workmen's Compensation Board
2 Bloor St. East,
Toronto, Ont. M4W 3C3
50 cents.

Occupational Diseases

This booklet gives a quick review of agents which may produce occupational diseases. It divides them into biological hazards, chemical hazards and physical agents, and discusses each in turn. It devotes much time to describing diseases associated with chemicals. This is good, as most of the exposure in industry is related to chemical hazards. However, the information, while useful, is scanty; giving the outcome of exposure, but not diagnosis or treatment. The booklet is oriented toward the WCB in that it starts off with a brief discussion on how to file a claim; however, even here the information is brief and does not mention such things as the forms to be completed by physicians.

In conclusion, this booklet is useful for an overview of occupational diseases and would be a good beginning for GPs who might want an introduction to occupational medicine.

G. Debow

News and Notes

Video Tapes

The following video tapes are available through the resource centres and may be viewed on request.

In Safe Hands

A brief introduction shows some of the accidents which can occur when fork truck drivers are untrained or careless. The film goes into the positive aspects of good fork truck driving. These are clearly presented to trainees by experienced instructors and are briefly interspersed with examples of accidents caused by not observing the rules.

21 minutes.

Colour.

Road Transport Training Board (Great Britain).

Making Light of Lifting

Reveals the secret of lifting objects of all sizes, shapes and weights, safely, easily and simply. How? By using the Kinetic Method, a scientific technique that enables people to make maximum use of their strength, yet avoids the stresses that lead to back injury.

17 minutes.

Colour.

Imperial Chemical Ind.

Construction Safety Series

Cost of Chaos – Building Trades Council, Great Britain.

Excavating – Building Trades Council, Great Britain.

Lifting Equipment – Building Trades Council, Great Britain.

Mechanical Aids – Building Trades Council, Great Britain.

Each of these short films shows how to prevent accidents in specific areas of construction. Each film illustrates a series of safety violations, then suggests using the “stop film” technique to allow time for discussion. The films are made in Great Britain; therefore, there are some points of legislation which are different.

These film stops can be used very effectively to discuss the difference between our legislation and theirs. When the film is resumed, it explains the violations and demonstrates the correct and safe way of doing things.

Each film 8 to 10 minutes.

Colour.

I am Joe's Lung

The lung is in many ways the most vulnerable organ of the human body. This film is an inside view of an average adult lung that shows what happens under normal conditions and why smog, dust and smoke can literally take your breath away.

25 minutes.

Colour.

Reader's Digest.

I am Joe's Spine

Through clever animation and the voice of Burgess Meredith, Joe's spine shows and tells us what's happening as Joe twists, bends, lifts, stretches and stoops in the process of cleaning out his attic. When the crunch comes, Joe's spine explains how future pain and problems can be avoided through proper exercise.

25 minutes.

Colour.

Reader's Digest.

Burn Emergency

Introduces the newest methods of caring for burn victims until they can reach a hospital or burn centre. The film shows how to care for heat burns, inhalation burns, electrical and chemical burns, and how to estimate burn damage.

25 minutes.

Colour.

San Francisco Fire Dept. and Mission Hospital S.F.

Don't Let Him Die

Deals in a dramatic way with emergency life-saving measures that the layman should know and can easily master. Updating techniques many of us learned a few years ago – and are now obsolete – the film covers heart failure, drowning, serious bleeding and shock.

16 minutes.

Colour.

Royal Navy.

Heads, You Lose

With unforgettable realism, this film zeros in on the big and little accidents that often make a construction site resemble a battle field. It then shows how most of this costly carnage could have been avoided simply and easily, just by using common sense, the first ingredient of safety.

20 minutes.

Colour.

National Federation of Building Trades (Great Britain).

Blasting Vibrations

This film gives the explanation of how explosives function. It shows the cause and effect of vibrations, which are shock and/or sound waves.

25 minutes.

Colour.

Canadian Industries Ltd.

Toxic Hazards in Industry

Some toxic hazards, ignored or recognized too late, have lead to great misery from diseases such as asbestosis, silicosis, and mercury poisoning. Today, with the multiplication of industrial processes and the utilization of more chemicals of greater complexity, the hazards have increased still further. This film sums up the responsibilities of management: to identify the hazards, to communicate them and to provide adequate protection; and of the work force: to take heed of the hazards and to use the protective equipment provided.

20 minutes.

Colour.

Imperial Chemical Ind.

Have a Good Day, Dear

This film on safety in an office environment points out that it is often our everyday habits that create the greatest hazards. It illustrates that both the obvious and not so obvious hazards can cause accidents with their resultant pain and expense.

18 minutes.

Colour.

Imperial Chemical Ind.

Is There Anything I've Forgotten?

Set in an industrial plant, this film illustrates the importance and seriousness of the checking and double checking procedures leading up to working in an enclosed space. It demonstrates the value of a formal procedures list. Even when rules are strictly adhered to, outside sources can still cause an accident.

20 minute videotape.

Colour.

Imperial Chemical Ind.

The Unplanned

An industrial accident prevention film illustrating two basic accident prevention tenets: that an accident usually has several causes, and that in most accidents, there is a difference between the accident causes and the injury cause. A situation is portrayed in a large machine shop, where a series of unsafe acts, combined with unsafe conditions, lead to an accident in which a man's arm is caught in an unguarded machine. A film to stimulate discussion among managers, supervisors and safety professionals, as well as for all industrial employees.

20 minutes.

Colour.

National Film Board of Canada for Labour Canada.

Your House in Order?

This film deals with the somewhat neglected subject – Industrial Housekeeping. As much as a third of all industrial accidents can be attributed either directly or indirectly to untidiness, slack working habits or to people who do not bother to think what their actions may do to others. The result is injury, lost time and considerable damage to plant and property. This film places the responsibility firmly where it belongs – on those who are most at risk – it makes the point that good housekeeping must involve everyone, including management.

20 minutes.

Colour.

Millbank Films Limited of England.

The Man in the Middle

The supervisor is the man in the middle. He is required to maintain full production, yet at the same time to look after the health, safety and welfare of his employees. Faced with the twin pressures of production and safety, where should his priorities lie? A film which will provoke much thought and discussion about attitudes to safety.

22 minutes.

Colour.

Millbank Films Limited of England.

Rubble Trouble

Industrial rubbish, carelessly stored, can become a breeding ground for fire. Oily rags, dust, trash and rubbish were born to burn. A carelessly tossed match, a spark of static electricity, or spontaneous combustion could result in a disastrous and costly industrial fire – resulting in the loss of valuable property, machinery and human life. This film shows how to prevent disaster by illustrating the most efficient methods of collecting, storing and removing industrial waste.

16 minutes.

Colour.

Pass & Seymour Inc.

Step Right Up

The correct procedures of ladder use are illustrated, including: maintenance, inspection, carrying, erecting and securing, ascending and descending. An excellent training film providing thorough coverage on the proper use of stepladders, straight ladders, and extension ladders. Over 25 different safety points are demonstrated.

19 minutes.

Colour.

Flammable Liquid Fire Safety

Correct methods of using, storing, and transporting flammable liquids are detailed. The film also suggests the use of fire warning devices and demonstrates the importance of designating a special storage area in the plant. It illustrates, dramatically, the significance of installing and maintaining automatic sprinkler systems.

18 minutes.

Colour.

Living with Dust

The film shows several ways of working to avoid excessive exposure to industrial dusts – containment, ventilation and personal protective devices.

15 minutes.

Colour.

3M Company.

Safe Handling of Compressed Gases

The film covers safety points in the handling and storage of compressed gases.

20 minutes.

Colour.

Metheson Gas Products.

Danger Zone . . . Your Back

While providing instruction, the film is directed toward the attitudes of employees, forcing them to recognize lifting, carrying, and pushing-pulling tasks and getting them to *want* to perform the tasks properly.

The film is divided into three sections which permits it to be used even in brief safety meetings. At the end of each section a question appears on the screen followed by black. If desired, the film may be stopped at this point to discuss the question raised and the material in the preceding section. Examples have been included from many industries to challenge viewers to learn the concepts rather than the specific task depicted.

19 minutes.

Colour.

VISUCOM Production Inc.

You and Office Safety

This award-winning film makes its strong points by using visual hyperbole and laughter to avoid a serious lecture. It leaves the viewer with the idea that safety in an office can be assured through the exercise of common sense and by thinking ahead.

9 minutes.

Colour.

Xerox.

Role of the Medical Department in Hearing Conservation

Demonstrates in real-life situations the elements of the Navy Medical Department's program of hearing conservation: Noise Measurement and Analysis, Engineering Control, Audiometry, Use of Protective Devices, and Education. Emphasizes prevention of hearing loss by means of periodic audiograms and the wearing of hearing-protection devices.

28 minutes.

Colour.

U.S. Navy.

Have You Heard?

A motivational and information film describing the hazards of high intensity noise found in military or industrial environments which can lead to progressive and permanent hearing loss. The film shows the effect hearing impairment can have in work and family relationships, illustrates the pathology of hearing loss from high intensity noise through the destruction of hair cells in the organ of Corti, and prescribes various noise control measures to reduce these hazards.

26 minutes.

Colour.

U.S. Navy.

Coming Events

September 1-4, 1980	International Environment and Safety Exhibition and Conference. <i>London, England</i>
September 3-5, 1980	2nd International Conference on Environmental and Occupational Toxicology of Nickel. <i>Swansea, Wales, U.K.</i>
September 8-12, 1980	International Symposium on Inhaled Particles. <i>Cardiff, Wales, U.K.</i>
September 17-19, 1980	Quebec/Ontario Occupational Health Physicians' Meeting. <i>Hotel Toronto, Toronto</i>
September 22-25, 1980	8th International Conference of Occupational Health in the Chemical Industry. <i>Tokyo, Japan</i>
October 1-3, 1980	International Committee on Occupational Mental Health. <i>Athens, Greece</i>
October 20-21, 1980	Occupational Exposure to Noise and Vibration: Effects, Measurement and Control. <i>Montreal, Canada</i>
October 20-24, 1980	Ontario Occupational Health Nurses' Annual Conference. <i>Ottawa</i>



**Ontario
Ministry of
Labour**

Occupational
Health and Safety
Division

*Occupational
Health Branch*

400 University Ave.
Toronto, Ontario
M7A 1T7

Telephone:
(416) 965-3610